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SIMULATION OF THERMAL TRANSIENTS DURING DEEP DIVING,(U)
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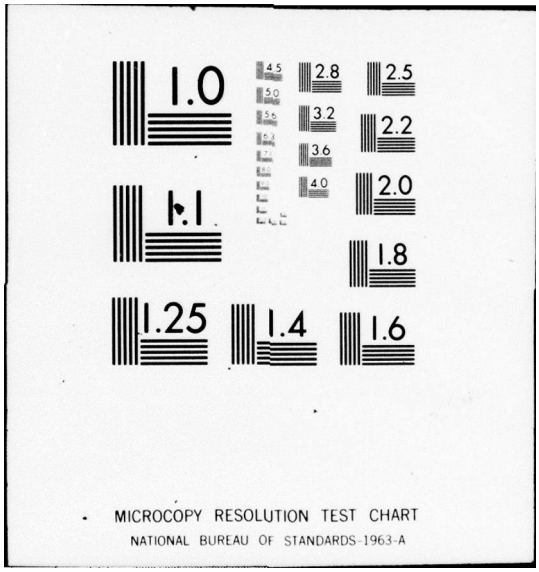
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This paper describes a mathematical model which has been developed to predict the thermal response of deep sea divers working at depths as great as 1000 feet. Under these conditions, divers are subjected to extreme thermal stress because the water temperature is approximately 0°C; the gas used for breathing and in the suit is helium which has a high thermal conductivity. Increased mass flow rate through the respiratory tract promotes heat loss when the breathing gas is not warmed. Since the experimental evaluation of new
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systems designed to protect divers is a difficult, time consuming process, it is worthwhile to have a reliable mathematical model for predicting diver performance under various conditions.

The mathematical model described in this paper subdivides the human into 15 cylindrical elements representing the head, thorax, abdomen, and proximal, medial, and distal segments of each arm and leg. Within each element the transient-state heat conduction equation is solved to obtain temperature profiles. Mass balances are also computed for oxygen, carbon dioxide and lactic acid. The program permits one to specify various kinds of wet and dry suits with either open or closed circuit heating.

Representative results are presented.

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SIMULATION OF THERMAL TRANSIENTS DURING DEEP DIVING

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INTRODUCTION

On June 5-6, 1975 a commercial diving team descended to a depth of 300 meters in the Bay of Laborador and helped to re-establish a drilling operation that had been interupted by a violent storm. While this was not a typical commercial diving job, it was indicative of things to come. Today, dives to depths of 200 meters are becoming commonplace in the North Sea, and leading commercial diving firms are developing systems for working at greater depths as drilling operations move further offshore.

Heat loss represents a serious problem for divers under these conditions. The severe thermal stress imposed on divers by immersion in cold water is compounded by factors related to the hyperbaric environment. At depths greater than 100 meters, the breathing gas is usually a mixture of helium and oxygen, which has a thermal conductivity approximately six times that of air. This causes a marked decrease in the thermal insulation of such garments as woolen underwear and foamed neoprene outer suits. Furthermore, closed cell foamed materials tend to compress and lose their insulating ability at depth. Heat loss through the respiratory tract also increases owing to an increase in gas density with increasing depth. If the breathing gas is not heated, heat loss through the respiratory tract will be approximately equal to the rate of metabolic heat generation at a depth of 200 meters, and since the ventilation rate is roughly proportional to the metabolic rate for moderate exercise levels, this will be true even when the diver is working (1).

Various systems have been developed to provide thermal protection for divers. At moderate depths commercial divers often wear a wet suit flooded with warm water supplied from the surface. Military divers engaged in relatively shallow clandestine missions must rely on self-contained systems, usually consisting of only a wet or dry suit without active heating. Chemical and electrical energy sources are under development for use when passive suits provide inadequate protection.

Experimental testing of such systems in hyperbaric chambers which approximate conditions encountered in the open sea is very expensive and time consuming. Therefore, it is desirable to have a mathematical model that can be used both to design new systems and to interpret test and field measurements. The purpose of this paper is to describe such a model and discuss a few results obtained using the model.

DESCRIPTION OF A HEAT AND MASS TRANSPORT MODEL FOR THE HUMAN

General Feathres

The physical system on which the equations of change are based is shown in Fig. 1. It consists of a number of cylindrical elements representing longitudinal segments of the head, trunk, arms, and legs. Each segment, consisting of a conglomeration of tissue, bone, fat, and skin, has a vascular system composed of three parts representing the arteries, veins, and capillaries. The circulatory path is faithfully reproduced in the sense that a tracer material introduced into an artery in a given segment will flow both into the capillaries of that segment and into the arteries of more distal segments. Blood leaving the capillary bed flows into a vein where it is mixed with venous blood from more distal segments. The mixed venous stream at the heart flows into the pulmonary artery; exchange of both heat and mass occurs in the lungs.

Within a given segment, physical parameters, such as density and specific heat, vary with radial position, while dependent variables, such as temperature, O_2 tension, metabolic rate, and blood perfusion rate, depend on both position and time. Certain variables, perfusion rate and metabolic rate, for instance, are defined by physiological control equations. Others, such as temperature and O_2 tension, are dependent variables whose immediate values depend on the previous history of the subject. Using this model one can start with specified initial conditions and follow changes that occur in response to various imposed conditions.

Equations of Change for Energy

Development of the heat transfer equations will be discussed in some detail. Since the equations for mass transfer are based on analogous concepts, they will be stated more concisely.

Heat which is generated by metabolic reactions is either stored in the element, carried away by circulating blood, or conducted to the surface where it is transferred to the environment. The basic equation for the thermal analysis is the heat conduction equation given below:

$$(\rho C_i)\frac{\partial T_i}{\partial t} = \frac{1}{r}\frac{\partial}{\partial r}\left(k_i r \frac{\partial T_i}{\partial r}\right) + h_{mi} + Q_{ci}(T_{ai} - T_i) + H_{ai}(T_{ai} - T_i) + H_{vi}(T_{vi} - T_i), \quad (1)$$

in which

$T_i(t,r)$ = instantaneous temperature of the tissue, bone, or viscera at a distance r from the axis of the i^{th} element,
$\rho_i(r)$ = density of tissue,
$C_i(r)$ = specific heat of tissue,
$k_i(r)$ = effective thermal conductivity of tissue,
$h_{mi}(t,r)$ = metabolic heat generation rate per unit volume,
$Q_{ci}(t,r)$ = product of the mass flow-rate and specific heat of blood entering the capillary beds per unit volume,
$H_{ai}(t,r)$ = heat transfer coefficient between arteries and tissue per unit volume,
$H_{vi}(t,r)$ = heat transfer coefficient between veins and tissue per unit volume,
$T_{ai}(t)$ = temperature of arterial blood,
$T_{vi}(t)$ = temperature of venous blood.

It should be observed that this form of the heat conduction equation is applicable only to an axially symmetrical system in which longitudinal conduction of heat is negligible. If the subject is moving so that there is a uniform flow of air or water around each of the elements, the analysis should apply. H. H. Pennes (2) has shown that longitudinal conduction in the arms is relatively unimportant, which should be true also in the legs (3), but not in the head.

Source terms on the right-hand side of Eq. 1 represent the rate of heat generation by metabolic reactions, the net rate at which heat is transferred from arterial blood to tissue, and the rate at which heat is transferred from venous blood to tissue. It is assumed that there is perfect heat transfer between blood in the capillaries and neighboring

tissue; i.e., the temperature of blood leaving a capillary bed is equal to the temperature of the neighboring tissue. As a first approximation, it is assumed also that the rate of heat transfer from blood in large vessels to neighboring tissue is proportional to the difference between the blood and tissue temperatures. The proportionality factors are called H_{ai} for arteries and H_{vi} for veins.

Since the temperature of blood in large vessels changes with time, it is necessary to write two more thermal energy balances. In formulating an equation for arterial blood, we assume that arteries in the i^{th} element form a pool having a uniform temperature, T_{ai}. The rate of accumulation of thermal energy in this reservoir is equal to the sum of the net rate at which heat is carried into the pool by flowing blood, the rate at which heat is transferred from neighboring tissue to blood in the pool, and the rate at which heat is transferred directly from the venous pool to the arterial pool owing to the proximity of certain arteries and veins. This equality is expressed mathematically by the following equation.

$$(MC)_{ai}\frac{dT_{ai}}{dt} = Q_{ai}(T_{am} - T_{ai}) + 2\pi L_i\int_0^{a_i} H_{ai}(T_i - T_{ai})rdr + H_{avi}(T_{vi} - T_{ai}), \quad (2)$$

in which

$T_{am}(t)$ = temperature of the blood entering the arterial pool,
M_{ai} = mass of the blood contained in the arterial pool of the i^{th} element,
C = specific heat of blood,
$Q_{ai}(t)$ = product of the mass flow rate and specific heat for blood entering the arterial pool,
L_i = length of the i^{th} element,
H_{avi} = heat transfer coefficient for direct transfer between large arteries and veins.

The corresponding equation for the venous pool is

$$(MC)_{vi}\frac{dT_{vi}}{dt} = Q_{vi}(T_{vn} - T_{vi}) + H_{avi}(T_{ai} - T_{vi}) + 2\pi L_i\int_0^{a_i}(Q_{ci} + H_{vi})(T_i - T_{vi})rdr \quad (3)$$

in which

$Q_{vi}(t)$ = product of mass and specific heat for venous blood flowing into the venous pool of the i^{th} element from the n^{th} element.

Since it is assumed throughout this analysis that M_{ai} and M_{vi} are constants,

$$Q_{ai}(t) = Q_{vi}(t) + 2\pi L_i\int_0^{a_i} Q_{ci}(t,r)rdr \quad (4)$$

The equation for the venous temperature in the abdominal section is slightly different from Eq. 3 because a vein from each leg flows into this section. It is also necessary to modify the equations for the thoracic section because all venous streams terminate and arterial streams originate in this section. It is assumed that the temperature of blood entering the pulmonary capillaries is equal to the "cup mixing" mean temperature of venous streams entering the right ventricle. This necessitates a change in Eq. 1 because the temperature of venous blood entering the pulmonary capillaries is different from the temperature of arterial blood entering more superficial capillaries of the thorax:

$$(\rho C)_1\frac{\partial T_1}{\partial t} = \frac{1}{r}\frac{\partial}{\partial r}\left(k_1 r\frac{\partial T_1}{\partial r}\right) + h_{m1} + Q_{ca}(T_{al} - T_1) + Q_{cv}(T_{vl} - T_1) + H_{al}(T_{al} - T_1) + H_{vl}(T_{vl} - T_1), \quad (5)$$

in which

$Q_{ca}(t,r)$ = product of the mass flow rate and specific heat for arterial blood flowing into capillaries,
$Q_{cv}(t,r)$ = product of the mass flow rate and specific heat for venous blood flowing into pulmonary capillaries.

Equations 2 and 3 must be modified to take cognizance of the fact that venous blood flows into the pulmonary capillaries which in turn empty into the arterial pool:

$$(MC)_{al}\frac{dT_{al}}{dt} = 2\pi L_1\int_0^{a_1} Q_{cv}(T_1 - T_{al})rdr + H_{avl}(T_{vi} - T_{al}) + 2\pi L_1\int_0^{a_1} H_{al}(T_1 - T_{al})rdr \quad (6)$$

$$(MC)_{vl}\frac{dT_{vl}}{dt} = \sum_i Q_{vli}(T_{vi} - T_{vl}) + H_{avl}(T_{al} - T_{vl}) - q_{rvl} + 2\pi L_1\int_0^{a_1} H_{vi}(T_1 - T_{vl})rdr \quad (7)$$

in which

$q_{rv_1}(t)$ = rate at which heat is transferred from venous blood in the thorax to air in the respiratory tract,
$Q_{vli}(t)$ = rate at which venous blood flows from the i^{th} element into the venous pool in the thorax = $Q_{ai}(t)$ for those elements which are connected to the thoracic segment.

The total rate of heat loss through the respiratory tract depends on the ventilation rate and the temperature and humidity of inspired air. In this analysis, it is assumed that 10 percent of the heat loss through the respiratory tract comes from the arterial pool in the head, 10 percent from the venous pool in the head, and 80 percent from the venous pool in the thorax.

Before these equations can be solved uniquely, certain constraining conditions must be specified. Some of these take the form of initial conditions which specify all temperatures at the instant the transient begins:

$$T_i(0,r) = T_{oi}(r) \quad (8)$$

$$T_{ai}(0) = T_{aoi} \quad (9)$$

$$T_{vi}(0) = T_{voi}. \quad (10)$$

Also needed are boundary conditions which relate the subject to his environment. These take the form:

$$-k\left(\frac{\partial T_i}{\partial r}\right)_{r=a_i} = H_i[T_i(t,a_i) - T_{ei}] + E_i, \quad (11)$$

in which

H_i = heat transfer coefficient,
T_{ei} = effective environmental temperature,
E_i = rate of heat loss by evaporation.

$$H_i = H_{ci} + H_{ri} \quad (12)$$

in which

H_{ci} = heat transfer coefficient for convection,
H_{ri} = heat transfer coefficient for radiation.

For unclothed regions of the body, the surface is skin; but for clothed regions, it is the outer surface of the

garment. Since physical properties are permitted to vary with radial position in this model, garments of considerable complexity can be analyzed. One can include a layer of gas or water between skin and the garment by assigning suitable values for the thermal conductivity, density, and specific heat. It is also possible to include an undergarment which contains tubing through which warmed or cooled liquid passes.

Equations of Change for Mass

Corresponding equations for mass transfer are similar to those presented above. For accumulation of a particular component in tissue and blood, we have the following equations.

$$\varepsilon_t \frac{\partial C_t}{\partial t} = \varepsilon_t R - F_{tb} \tag{13}$$

and

$$\varepsilon_b \frac{\partial C_b}{\partial t} + q \frac{\partial C_b}{\partial x} = F_{tb} \tag{14}$$

in which

ε_t and ε_b = volumetric fractions of tissue and blood, respectively,
C_t and C_b = concentration in tissue and blood, respectively,
q = flux of blood through the capillaries in a unit area,
x = distance measured along a capillary,
F_{tb} = rate of exchange between tissue and capillary per unit volume,
R = rate of production per unit volume.

Although the basic equations for all components have the same form, the approximations which are appropriate in dealing with the distribution of material between tissue and blood depend on the particular component involved.

When dealing with oxygen, it is assumed that equilibrium exists between oxygen in myoglobin and hemoglobin. Typical functional relationships between oxygen content and oxygen tension for myoglobin and hemoglobin are shown in Fig. 2. Since myoglobin is essentially saturated at oxygen tensions greater than 20 Torr, the amount of oxygen stored in myoglobin remains constant under normal conditions. These stores are released when the oxygen tension falls below 20 Torr. The dissociation curve for myoglobin can be approximated by the dashed curve without introducing appreciable error. Then equilibrium between oxygen in myoglobin and hemoglobin implies that

$$C_{O_2 t} = 0.24\, C_{O_2 b}, \text{ if } 0 \le C_{O_2 b} \le .046 \text{ volume fraction}$$

$$= 0.011 \quad \text{if} \quad .046 < C_{O_2 b}. \tag{15}$$

When Eqs. 13 and 14 are added, one obtains

$$(0.24\, \varepsilon_t + \varepsilon_b) \frac{\partial C_{O_2 b}}{\partial t} + q \frac{\partial C_{O_2 b}}{\partial x} = \varepsilon_t R$$

$$\text{if} \quad 0 \le C_{O_2 b} \le .046 \tag{16}$$

or

$$\varepsilon_b \frac{\partial C_{O_2 b}}{\partial t} + q \frac{\partial C_{O_2 b}}{\partial x} = \varepsilon_t R \quad \text{if} \quad .046 < C_{O_2 b}.$$

In this case, R is a negative quantity which accounts for depletion of oxygen by metabolic reactions.

Allowance is made for the reduction in oxygen tension along a capillary by subdividing it into four sections, each treated as a well-mixed volume. Then Eq. 16 assumes the form

$$\frac{(0.24\, \varepsilon_t + \varepsilon_b)}{4} \frac{dC_{O_2,i}}{dt} + q\,(C_{O_2,i} - C_{O_2,i-1}) = \frac{\varepsilon_t}{4} R_i$$

$$\text{if } 0 < C_{O_2,i} \le .046 \tag{17}$$

or

$$\frac{\varepsilon_b}{4} \frac{dC_{O_2,i}}{dt} + q\,(C_{O_2,i} - C_{O_2,i-1}) = \frac{\varepsilon_t}{4} R_i$$

$$\text{if } .046 < C_{O_2,i}.$$

The subscript b has been dropped, but it is to be understood in the remainder of the paper that $C_{O_2,i}$ refers to the oxygen content of blood in the i^{th} segment of the capillary $(1 \le i \le 4)$. For the first segment, $C_{O_2,i-1}$ refers to the oxygen content of arterial blood entering the capillary.

Whenever the maximum rate of oxygen transport to muscle exceeds the demand, the rate of utilization is determined by the demand. During vigorous exercise, however, demand exceeds supply and anaerobic reactions occur. If C_{O_2} falls below a critical value in any capillary segment, it is assumed that the oxygen supply is inadequate and anaerobic glycolysis occurs with the production of lactic acid. The critical oxygen concentration was arbitrarily set at a constant value of 7.5 volume percent although it should depend on the density of capillaries and the local metabolic rate, both of which change during exercise.

Since the set of reactions involved in metabolism is very complex, we found it necessary to adopt a greatly simplified metabolic scheme which does, nevertheless, include several of the more important factors. Aerobic synthesis of ATP using glucose as the substrate proceeds according to the following overall reaction.

$$C_6H_{12}O_6 + 6\, O_2 + 38\, ADP + 38\, P_i$$

(glucose)

$$\rightarrow 6\, CO_2 + 6\, H_2O + 38\, ATP \tag{18}$$

In the absence of oxygen, synthesis of ATP proceeds as follows.

$$C_6H_{12}O_6 + 2\, ADP + 2\, P_i$$

(glucose)

$$\rightarrow 2\, C_3H_6O_3 + 2\, ATP \tag{19}$$

(lactic acid)

Lactic acid produced in muscle is transferred to blood in the capillaries and distributed throughout the body. It can be converted to glycogen in the liver according to an aerobic mechanism which oxidizes one mole of lactic acid to carbon dioxide and water for every four moles converted into glycogen, or it can be catabolized in muscle.

We assume that glucose is always available at the rate required. Hence, it is only necessary to account for three components - oxygen, carbon dioxide, and lactic acid. Specification of the rate of energy release assuming that only aerobic processes are involved defines the demand for oxygen. If the rate of oxygen supply as defined above is not adequate to meet the demand, the deficit will be provided by anaerobic reactions. However, 19 moles of glucose are consumed in the anaerobic production of the same amount of ATP that is produced using one mole of glucose in the aerobic process. Knowing this one can compute production rates for carbon dioxide, lactic acid, and heat for given values of demand and supply.

Equations of change for carbon dioxide are derived using the same approach. It is assumed that equilibrium exists between the carbon dioxide stores in tissue and blood, and that at equilibrium the concentration in tissue is one-half of the concentration in blood (4). The equation for

CO_2 which corresponds to Eq. 17 for O_2 is

$$\frac{(0.5\,\varepsilon_t + \varepsilon_b)}{4}\frac{dC_{CO_2,i}}{dt} + q\,(C_{CO_2,i} - C_{CO_2,i-1}) = \frac{\varepsilon_t}{4} R_{CO_2,i} \tag{20}$$

According to the simplified reaction scheme presented above,

$$R_{CO_2,i} = -R_{O_2,i}\ R \qquad \text{(in muscle)}$$

$$= -R_{O_2,i}\ R + 0.25R_{LA.G} \qquad \text{(in liver)} \tag{21}$$

in which $R_{LA.G}$ = rate at which lactic acid is converted to glycogen in the liver

R = respiratory quotient $\cong$ 0.9.

Equations of change are required also for the lactic acid produced during anaerobic glycolysis. In this case, equilibrium does not exist between lactic acid in muscle and blood. Therefore, we assume that lactic acid is transferred from muscle to blood at a rate which is proportional to the difference in concentration; i.e.,

$$F_{tb} = D_{LA}\,(C_{LA_t} - C_{LA_b}) \tag{22}$$

Equations 13 and 14 reduce to the following forms:

$$\frac{\varepsilon_t}{4}\frac{dC_{LA_t,i}}{dt} = \frac{\varepsilon_t}{4} R_{LA,i} - D_{LA}(C_{LA_t,i} - C_{LA_b,i}) \tag{23}$$

and

$$\frac{\varepsilon_b}{4}\frac{dC_{LA_t,i}}{dt} + q(C_{LA_b,i} - C_{LA_b,i-1}) = D_{LA}(C_{LA_t,i} - C_{LA_b,i}) \tag{24}$$

The net rate of production of lactic acid, $R_{LA,i}$, is defined as follows:

$$R_{LA,i} = R_{mi} - R_{O_2,i} \qquad \text{(in muscle)}$$

$$= -1.25\ R_{LA.G,i} \qquad \text{(in liver)} \tag{25}$$

in which $R_{LA.G,i} = k_{LA} C_{O_2,i}\ C_{LA_t,i}$. (26)

Values for the constants, D_{LA} and k_{LA}, are not available in the literature; they must be estimated by comparing computed results with a small number of reported experimental observations.

Equations of change for inert gases are also easily obtained. Again it is assumed that local equilibrium exists between gas dissolved in tissue and blood. Because there is no production, Eqs. 13 and 14 reduce to

$$(r\,\varepsilon_t + \varepsilon_b)\frac{dC_i}{dt} + q\,(C_i - C_{i-1}) = 0 \tag{27}$$

in which r = ratio of the solubilities in tissue and blood.

In the lungs additional terms are required to account for exchange of gases between pulmonary capillaries and the alveolar space. Metabolic reactions are neglected in the lungs, and we have for both reactive and inert gases

$$(r\,\varepsilon_t + \varepsilon_b)\frac{dC_i}{dt} + q\,(C_i - C_{i-1}) = -F_{ba,i} \tag{28}$$

in which $F_{ba,i}$ = rate at which the component is transferred from blood to alveolar gas.

Since q for the lungs is a relatively large quantity, one can neglect accumulation of mass in the capillaries and surrounding tissue and approximate Eq. 28 by

$$q(C_a - C_4) = -F_{ba} \tag{29}$$

in which C_a = concentration in the pulmonary artery, F_{ba} = total rate of transfer along a capillary. The method used in evaluating F_{ba} will be discussed later.

In developing equations of change for large arteries and veins, it is assumed that there is neither reaction nor exchange of mass with surrounding tissue. Hence, we have the following equation for oxygen in the arterial pool of the $i^{\underline{th}}$ element.

$$V_{ai}\frac{dC_{O_2a,i}}{dt} = q_{ai}\,(C_{O_2a,m} - C_{O_2a,i}) \tag{30}$$

in which $C_{O_2a,i}$ = oxygen concentration of arterial blood in the $i^{\underline{th}}$ segment,

$C_{O_2a,m}$ = oxygen concentration of blood entering the the arterial pool,

V_{ai} = volume of the arterial pool,

q_{ai} = volumetric flow rate of blood entering tne arterial pool.

The corresponding equation for oxygen in the venous pool is

$$V_{vi}\frac{dC_{O_2v,i}}{dt} = q_{vi}\,(C_{O_2v,n} - C_{O_2v,i}) + 2\pi L_i\int_0^{a_i} q(C_{O_2,4} - C_{O_2v,i})rdr \tag{31}$$

in which q_{vi} = volumetric flow rate for venous blood entering the $i^{\underline{th}}$ element from the $n^{\underline{th}}$ element, and the term $C_{O_2,4}$ under the integral refers to the oxygen concentration of blood flowing into the venous pool from capillaries of the element.

Corresponding equations for other components can be obtained by merely changing the identifying subscript in Eqs. 30 and 31.

Heat and Mass Transport in the Lungs

When evaluating exchange rates in the respiratory system, we assume that the lungs can be treated as a well-mixed region of constant volume, V_L, through which gas passes at a rate, V_E. Since heat and water vapor are transferred with ease in the lungs, it is assumed that expired air is saturated with water vapor at the temperature of the respiratory tract, T_r, defined as follows (5):

$$T_r = 24.4 + 0.32\ T_i \tag{32}$$

in which T_i = temperature of inspired gas, °C. The rate at which sensible heat is lost through the respiratory tract is easily computed as follows

$$Q_{sr} = V_E\,\rho_g\,C_p\,(T_r - T_i) \tag{33}$$

The corresponding rate of latent heat loss is

$$Q_{lr} = V_E\frac{\Delta H_v}{22.4}\left(\frac{P_{H_2O} - P_{H_2O,i}}{760}\right)\left(\frac{273}{273+T}\right) \tag{34}$$

in which ΔH_v = molar latent heat of vaporization of water.

Other gases are exchanged also across the walls of the pulmonary capillaries. Since this process for a given gas is limited by the rate at which gas is released in the capillaries, concentration changes in the lungs depend on cardiac output as well as the ventilation rate. For O_2 the equation of

change can be written in the form

$$V_L \frac{dP_{O_2}}{dt} = 860F_{ba,O_2} + V_E(P_{O_2,i} - P_{O_2}) \quad (35)$$

in which $P_{O_2,i}$ and P_{O_2} are the partial pressures of oxygen in inspired and expired air, respectively. The numerical factor, 860, appears because gas concentrations in blood are measured in ml of dry gas at STP/ml of blood; a common factor, 8-47 (B = pressure), would normally appear in the denominator of each term. Corresponding equations for other components can be written by merely changing the subscript designating the gas.

Following common practice we assume that the partial pressure of each component in the alveolar gas is equal to the tension of that gas in blood as it leaves the pulmonary capillaries. Strictly speaking equality will not exist because a difference in pressure is required to drive the exchange, but the difference is usually negligible. Our analysis would not be complicated unduly by using a more precise expression. We assume also that 2% of the pulmonary blood flow is shunted (6).

Evaluation of the mass transfer rate between blood in the pulmonary capillaries and alveolar gas requires knowledge of the gas tension in blood. The oxygen dissociation equation and carbon dioxide buffer equations developed by Grodins, Buell, and Bart (7) are used in this paper. Both the Haldane and the Bohr effects are included in this model. The bicarbonate concentration decreases as lactic acid concentration increases (8). The discrepancy between the concentration of oxyhemoglobin predicted using this model and generally accepted values is not great.

PHYSIOLOGICAL CONTROL EQUATIONS

Feedback Control

The equations of change presented above permit one to compute temperatures and concentrations when physiological parameters, such as local metabolic rates, tissue perfusion rates, and the ventilation rate, are known. These rates, in turn, depend on temperature, oxygen and carbon dioxide tensions, lactic acid concentration, and level of exercise. Since the formulation of control equations is still an active area of research, this portion of the model is necessarily somewhat incomplete.

Considerable progress has been made recently in obtaining a quantitative description of thermal regulatory mechanisms, especially under warm conditions (9, 10). Using concepts drawn from the theory of feedback controllers, one separates the control system into three parts. The first contains various thermoreceptors which define the thermal state of the system. The second consists of the control center which receives information from the receptors, integrates it, and transmits appropriate commands to the effector systems, which constitute the third part. Effector commands may be modified depending on local conditions before being translated into effector action.

Thermoreceptors have been identified by either observing the effect of a given temperature change on quantities such as sweat rate and skin blood flow rate, or by observing the change in electrical activity in nerve fibers as the temperature changes. Areas in which thermosensitivity has been demonstrated include the preoptic area and the anterior hypothalamus in the brain, the abdominal viscera, and the skin. Other areas have been suspected, but since it is extremely difficult to restrict thermal stimulation to a specific area, the function of a given area is not easily evaluated.

The output from each thermoreceptor is generally defined in terms of an "error signal" which is the difference between the instaneous temperature and the reference temperature, or set-point, for a given area. One can question the existence of a large number of setpoints, but an alternative point of view is that this form of the receptor equation represents a linearization of more complex equations about an equilibrium state. Some of the more recent work (11) includes a rate-of-change term in the error expression, but we have not included that in the present model. Positive errors are assumed to represent outputs from warm receptors while negative errors represent outputs from cold receptors.

Peripheral afferents from receptors in the skin are integrated by forming weighted sums of the signals coming from warm and cold receptors.

$$S_{aw} = \sum_{i=1}^{15} F_i(T_{skin,i} - T_{set,i}) \quad (36)$$

$$S_{ac} = \sum_{i=1}^{15} F_i(T_{set,i} - T_{skin,i})$$

in which S_{aw} and S_{ac} are the integrated warm and cold signals, respectively; F_i is the weighting factor for the i^{th} element; only positive terms are included in the sum. Effector signals depend on the integrated peripheral afferent signals and the error signal generated in the brain.

Control Functions for Perfusion

One mechanism available for regulation of central temperature is control of the rate at which blood perfuses the skin. As deep body temperatures rise, blood flow to the skin increases, thereby increasing skin temperature and the rate of heat transfer to the environment. When circumstances require conservation of heat, subcutaneous capillaries constrict reducing the rate at which heat is brought to the surface by convection. These responses are incorporated into the model through two variables, DIL and CON, which are the increase in cardiac output owing to increased perfusion of the skin and an index of the increase in peripheral circulatory resistance.

$$DIL = 1.95(T_{brain} - T_{set,brain}) + 0.125\ (S_{aw} - S_{ac}) \quad (37)$$

$$CON = 5(T_{set,brain} - T_{brain}) + 5(S_{ac} - S_{aw}) \quad (38)$$

DIL is measured in liters/min and CON is dimensionless. The blood flow rate to each region is computed by adding a fraction of DIL to the base flow rate for the region and dividing the resulting value by $1 + FC_i$ CON, in which FC_i is a constant factor defined for the region. It is assumed also that local factors cause a doubling or halving of the perfusion rate for each 6°C change in the local temperature. Hence, the final expression for the cutaneous blood flow rate to the i^{th} element contains a multiplicative factor, $\text{Exp}\,[(T_{skin,i} - T_{set,i})/8.66]$. Although the gross responses described by the model have been verified through measurement of the mean thermal conductance under various conditions, one should not conclude that our state of knowledge is complete, or even generally satisfactory.

For example, the model does not describe the observations of Spealman (12) who measured directly the blood flow rate to the hand immersed in water at various temperatures and showed that the perfusion rate declines with declining bath temperature to 10°C. Below this temperature there is a striking increase in the hand blood flow rate, which frequently attains values comparable to those observed in a water bath at 35°C. If the subject is comfortably warm except for the hand, alternating periods of vasodilation and vasoconstriction (hunting) occur. When the subject is chilled, the increase in blood flow is not so pronounced. If the subject is uncomfortably warm, the blood flow rate to the hand remains high even in water at 10°C.

The perfusion rate used in the model for muscle depends on the local metabolic rate and the end-capillary oxygen tension. When $P_{O_2} < 45$ Torr, the resting value is multiplied by a factor $1 + (45 - P_{O_2})/8$. During exercise a term which is proportional to the difference between the metabolic rates in the working and resting states is added to the resting blood flow rate. It is assumed that perfusion rates do not change instantaneously, but instead change exponentially with a time constant of 30 seconds for all tissue except brain which has a time constant of 0.3 seconds.

During vigorous exercise blood flow to inactive muscle is

reduced in order to meet the demand of active muscles without exceeding limitations on cardiac output (13, 14). This feature was incorporated into the model by multiplying each flow rate for muscle by a factor 1 - 0.00032 exp (0.32 cardiac output); cardiac output is measured in liters/min. Since cardiac output represents the summation of local perfusion rates, its evaluation requires the solution of a transcendental equation.

Arterial-venous oxygen difference can be used to ascertain the blood flow rate for regions which are supplied by an accessible artery and are drained by a representative vein. In this way it has been established that the cerebral blood flow rate varies considerably, apparently in response to varying arterial carbon dioxide tension (6). Experimental data reported by Lambertsen et al (15, 6) were used to define the relationship which is used in the model to compute the rate of cerebral blood flow. Furthermore, when the end-capillary oxygen tension falls below 45 Torr, the base flow rate is multiplied by $1 + (45 - P_{O_2})/45$. We assume also that high arterial oxygen tensions ($P_{aO_2} > 500$) reduce the cerebral blood flow rate by a factor $1 - 0.00005\ (P_{aO_2} - 500)$.

Admittedly, the perfusion control equations incorporated into the current model represent only a first approximation to reality. More precise relationships, such as those presented by Rowell (13), should be built into the model and the resulting effect on system performance should be evaluated.

Control Functions for Sweating

Man's ability to regulate his central temperature by controlling only cutaneous perfusion is limited to a rather narrow range of environmental conditions and exercise levels. When heat must be transferred to the environment at higher rates than permitted by convection and radiation alone, the subject begins to sweat. Since sweating is such an important thermoregulatory mechanism and one that can be observed directly, numerous studies have been reported in the literature. Nevertheless, important questions remain unanswered.

One concerns the importance of a non-thermal component in the forcing function for sweating during exercise. Van Beaumont and Bullard (16) provided evidence in support of such a component with their observation that an increase in the sweat rate occurs within a few seconds of the onset of exercise by a subject who is already sweating. The increase in sweat rate occurs prior to a significant rise in any body temperature. Nadel et al (17) report that the rapid increase in sweating is transient, persisting only for the first minute or two of exercise. Thereafter, the rate of sweating seems to be described adequately by a proportional feedback control mechanism. The efferent outflow, S_w in Kcal/min, is computed as follows

$$S_w = 5.33(T_{brain} - T_{set,brain}) + 0.48(S_{aw} - S_{ac}) \qquad (39)$$

A fraction, F_i, of the efferent outflow is added to the base evaporation rate of the i^{th} element, and the result is multiplied by the factor $\exp[(T_{skin,i} - T_{set,i})/10]$ to determine the local rate of sweating.

The rate of evaporation may be smaller than the rate of sweat production if the environmental humidity is too high. Furthermore, it appears that water standing on the skin inhibits the secretion of sweat (18), but this effect is not included in the model.

Control Functions for Shivering

Although shivering can be measured easily, the control function for shivering has remained ill-defined because steady-state measurements are difficult to obtain. However, it seems likely that both the skin and central temperatures must fall below their threshold levels before shivering can occur. Experimental studies (19) appear to be described adequately by a multiplicative control function of the form

$$S_h = 0.35(T_{set,brain} - T_{brain})S_{ac} \qquad (40)$$

S_h is measured in Kcal/min. The increase in metabolic rate of the i^{th} element owing to shivering is calculated by multiplying Sh by a fraction, FCH_i.

Control Equations for the Ventilation Rate

Since breathing frequency and tidal volume are relatively easy to measure, one might expect that a generally applicable control equation would be available for ventilation. However, this is not the case because the primary sensors which affect ventilation are inaccessible in man. It is known that receptors are located in the aortic and carotid bodies, as well as in the brain. These receptors are stimulated by changes in both pH and the CO_2 tension of arterial blood perfusing the region. Since changing CO_2 tension also changes pH, it is difficult to ascertain whether the receptors are responding to the change in CO_2 tension or hydrogen ion concentration. The peripheral receptors also respond to large changes in O_2 tension; ventilation is stimulated by hypoxia and depressed by O_2 tensions in excess of 1 atmosphere. Another complicating factor is that cerebral perfusion rate is affected by changes in O_2 and CO_2 tensions. Furthermore, there exists a drive of undetermined origin which causes a prompt increase in the ventilation rate at the beginning of exercise.

Lambertsen (6) has presented an excellent discussion of factors which must be considered in developing a control model for ventilation. His view of ventilatory control is based in part on an interpretation of step response data. A thorough discussion of these experiments was presented recently by Gelfand and Lambertsen (20), who concluded that three sensors contribute to the overall response. A fast component, which has a delay time of 6-8 sec and a time constant of 5 sec was assigned to the peripheral sensor. Two components, which have delay times of 16-18 sec and time constants of 10 to 100 sec, were assigned to sensors located in the central nervous system.

Further support for the "dual center hypothesis" is provided by measurement of the steady-state ventilatory response to a change in blood pH produced by infusion of fixed acids and bases while CO_2 tension is held constant. These experiments (21) show that an acid or base induced change in pH produces approximately 50 percent of the ventilatory response produced by an equal CO_2 induced change in pH. Lambertsen feels that both centers respond to $[H^+]$, rather than P_{aCO_2}, but one center lies behind a blood-brain barrier which $^{aCO}2$ transmits CO_2 and not H^+, while the other center is responsive to blood ^{2} $[H^+]$. Although the magnitude of the change is smaller, the ventilatory response to a step change in $[H^+]$ with P_{aCO_2} held constant is just as rapid as the response to a $^{aCO}2$ step change in P_{aCO_2}. The center which responds to $[H^+]$ must be very well ^{2} perfused and lack an appreciable diffusion barrier for ions.

Several attempts have been made to stimulate the system by a sinusoidally varying alveolar CO_2 tension. The most impressive effort was reported by Swanson and Bellville (22), who recognized that as the frequency increases, the relative importance of the peripheral receptors increases also. In this way, they were able to differentiate between responses generated by the peripheral and central receptors.

Recent papers by Stoll (23), Bellville, et al (24), and Swanson and Bellville (22) present independent sets of frequency response data which are in good agreement with each other. When one computes the frequency response function using the three-component dynamic model proposed by Gelfand and Lambertsen, it is found that the predicted and computed amplitude ratios are in reasonable agreement, but the predicted phase lag is much greater than the observed lag. The large predicted phase lag is a consequence of the 16-18 second delay time assigned to the two central receptors; a delay time of 6 seconds would have produced much better agreement. No obvious explanation for the discrepancy between the conclusions based on step response and frequency response data is available.

Recently Milhorn and Reynolds (25) have used "exponential peeling" to analyze transient response data taken in their laboratory. Their experience with this technique, which is the one used by Gelfand and Lambertsen, led them to question the validity of the control scheme proposed by Lambertsen (6). Although it is impossible at the present time to resolve conclusively the differences between models proposed by various investigators, the situation is not hopeless. There is generally good agreement about the steady-state response to an increase in CO_2 partial pressure. Since various investigators also agree about the nature of the slowly responding sensors, it is possible to construct a

respiratory controller which describes most commonly occurring transients with acceptable accuracy. The controller incorporated into the present model is based on Lambertsen's work (6), but other models could have been used.

It is hypothesized that three hydrogen ion concentrations are important. One is sensed by a peripheral receptor which is exposed to arterial blood. The other two are central receptors, one of which is exposed to blood while the other lies behind a barrier that is permeable to carbon dioxide but not hydrogen ions. We assume that the second central receptor responds as a first-order system, i.e.,

$$\frac{dP}{dt}_{CS,CO_2} = k_{CS} (\bar{P}_{CO_2} - P_{CS,CO_2}) \quad (41)$$

in which P_{CS,CO_2} = CO_2 tension at the central sensor site,

$\bar{P}_{CO_2}$ = mid-capillary CO_2 tension and,

k_{CS} = a constant = 1/25 sec.$^{-1}$.

The three hydrogen ion concentrations are weighted as follows to form a single control variable, CVE.

$$CVE = 0.12\ C_{a,H^+} + 0.44\ C_{v,H^+} + 0.44\ C_{CS,H^+} \quad (42)$$

C_{a,H^+} = arterial concentration in the thorax $\cong$ concentration in the head, and

C_{v,H^+} = end-capillary concentration in the brain.

The relationship between $V_{E,f}$ and CVE is shown in Fig. 3.

It is known that under certain conditions, the minute volume is influenced also by the arterial oxygen tension. The value obtained from Fig. 3 is modified as follows to form $V'_{E,f}$.

$$V'_{E,f} = V_{E,f} - COER(V_{E,f} - 6) \quad (43)$$

in which $COER = 0.1(P_{aO_2} - 104)/656$. If $P_{aO_2} < 37$, another term is added to $V'_{E,f}$.

$$V''_{E,f} = V'_{E,f} + 0.56 \exp [0.23(37 - P_{aO_2})] \quad (44)$$

These equations apply to resting subjects. During exercise V_E exceeds $V''_{E,f}$ by a considerable amount, presumably due to the influence of neurogenic factors (26). A change in central temperature also affects the ventilation rates

SOLUTION OF THE EQUATIONS

The equations described in the previous section were solved numerically using finite-difference techniques. This procedure provides considerable latitude in dealing with parameters and variables, such as thermal conductivity, specific heat, metabolic rate and perfusion rate, which vary with position and time. Each of the 15 major elements is subdivided into not more than 8 concentric regions, in each of which the properties are uniform. A representative arrangement is shown in Fig. 4. By properly defining these regions and assigning physical properties, one can simulate many different kinds of thermal systems.

Auxilliary programs and subroutines have been written to assist with the evaluation of physical properties. Since one objective of this study has been to evaluate the accuracy of the model by comparing computed results for a specific experiment with the corresponding experimental results, it was helpful to prepare an auxilliary program which could generate geometric values and physical properties for a specific individual. Input variables for this program include the subject's height, weight, percent fat, and the length, perimeter, and coronal and sagittal diameters of each of the major elements. Quantities such as the percentages of bone, blood, and viscera are taken from published tables. Output variables, which are saved in a file that can be read by the main program, include the locations of the radial nodes for each of the elements, identification of the boundary points which separate adjacent property regions, and specification of mean properties for each of the regions out to, and including, the skin.

A subroutine is available for evaluating the thermal properties of various kinds of outer garments, including a shirt, socks, trousers, shorts, dry suit with woolen underwear, and a wet suit which may be supplied with warm water. When a gas is involved either internal or external to the garment, one can specify whether it is helium or air. In the case of a closed cell foamed neoprene garment, the thermal conductivity, density, specific heat, and thickness are adjusted for pressure.

In solving the heat conduction equation numerically, one has a certain amount of freedom in formulating the finite-difference equations. We chose to use an implicit set of difference equations of the Crank-Nicholson type, which required the simultaneous solution of some two hundred and fifty equations at each time step. Although the coefficient matrix did not have the classical tridiagonal form that one customarily obtains, the set of equations was still well-conditioned for solution by Gaussian elimination. Readers who are interested in the details of the solution can find a fairly complete discussion in a previously published paper (27).

REPRESENTATIVE RESULTS

The number of cases that can be analyzed using this program is nearly infinite. Results for three typical cases will be discussed in this section.

Exposure to Elevated Temperatures in a Hyperbaric Chamber

It is widely recognized that divers are subject to chilling in a hyperbaric chamber containing helium. However, the equally great danger posed by an elevated chamber temperature was not fully appreciated until two divers perished in Scotland from accidental hyperthermia. Therefore, this is a worthwhile case to analyze. Two sets of computations will be discussed - one for a one hour exposure to 1 ata air at 41°C and the other for a similar exposure to 7 ata helium (corresponding to a depth of 62 meters). In each case the diver is dressed in a short sleeved shirt, cotton trousers, and shoes.

Shown in Fig. 5 are graphs of the rectal and mean skin temperatures for these two exposures. In air the diver's mean skin temperature rises from 34.2°C to 35.4°C during the first six minutes of exposure. Then profuse sweating occurs and evaporative cooling lowers the skin temperature to a reasonable level. During the entire exposure, the diver receives more heat by convection than he loses by evaporation, and the rectal temperature increases from 37.2°C to nearly 38°C. While this would be an unpleasant experience, it would not be damaging to health.

The corresponding graphs for exposure to helium indicate that this condition is potentially dangerous. Except for a short pause in the rising skin temperature, both temperatures increase steadily during the entire exposure. Several factors contribute to the severity of the helium exposure. One, of course, is that the thermal conductivity of helium is six times that of air which enhances the rate at which heat is transferred to the skin by convection. Another is that the greater density of gas that is breathed promotes heat gain through the respiratory tract. The third factor, which is not so obvious, is that the diffusivity of water vapor decreases with increasing gas density. This means that the maximum rate of evaporation decreases as the pressure increases, and sweating becomes ineffective as a means for coping with rising temperatures.

Evaluation of a Commonly Used Wet Suit

Wet suits made from closed cell foamed neoprene are often used to provide thermal protection during relatively shallow dives. A typical ensemble consists of 1/4 in. thick "Farmer John" pants and jacket with hood, a 1/8 in. thick vest with hood, 1/4 in. thick soft-soled boots, and three-fingered mittens. A face mask and fins are also worn. The thermal response of a diver of medium build while wearing this suit for two hours in water at 4, 16, and 27°C was studied at 1 ata. In each case, the diver alternated periods of rest with periods of moderate swimming as shown in Figs. 6 through 8.

It appears that this suit is quite adequate for use in 16°C water. There is a modest decrease in rectal temperature during the resting period, but the trend is reversed during exercise. It is interesting to note that there is an undershoot following the end of the rest period and an overshoot following the exercise period. The rate of gas consumption during the exercise periods is approximately double the

resting rate. As the dive progresses, there is a long term trend toward increasing gas consumption, which can be attributed to low level shivering caused by the decrease in rectal and skin temperatures. Nevertheless, it appears that the diver is quite comfortable under these conditions.

Corresponding graphs obtained when the water temperature is 4°C indicate that the suit does not provide adequate protection for long periods of exposure at this temperature. The rectal temperature falls 1°C during the first 30 minutes of the dive. This results in vigorous shivering with a corresponding increase in the rate of gas consumption. Some divers, especially those who are somewhat fatter than the model diver, could tolerate this exposure for an extended period of time, but they would be quite uncomfortable and fatigued at the conclusion of the dive.

A third set of computations was performed for 27°C water. The results are shown in Fig. 8, where one can see that the diver overheats during the exercise period. From these results one can conclude that this suit is ideal for shallow dives when the water temperature is approximately 16°C.

The effect of depth on performance while wearing this suit is illustrated in Fig. 9 where graphs are presented for a 30 meter dive in 16°C water. It is obvious that the suit is very inadequate under these conditions. Violent shivering occurs early in the dive, and it is doubtful whether the diver could remain in the water very long. The flat gas consumption rate during exercise occurred because of an arbitrary maximum value specified in the program.

The 1 ata results are consistent with observations made at the Naval Coastal Systems Laboratory in Panama City, Florida (28). When the wet suit used in these calculations was tested, it was found that the suit was ideal for use in 16°C water. At 4°C shivering occurred and many divers were unable to complete a 6 hour dive. The 27°C dives were conducted wearing a partial suit to avoid overheating. Hence, the model does appear to be capable of simulating reasonably well the performance of divers under these conditions.

Use of a Liquid Heated Dry Suit

Prolonged dives to depths below 200 meters require the use of auxiliary heating to keep the diver comfortable. Although they have not been used to date, closed-circuit liquid heated suits would appear to be more efficient than the open-circuit suits that are generally used. Therefore, several cases were analyzed to determine whether the closed-circuit concept is reasonable.

In the present version of the model, a liquid heated garment can be defined by specifying the heat transfer coefficient and area of contact for each element of a suit such as the one illustrated in Fig. 10. Since the fluid which serves as the heat source may flow serially past several heat exchange areas, it is necessary to specify the flow path for each stream. This is done by indexing each segment of the path and developing a table in which are specified (1) the anatomical element with which each exchanger segment is in contact, (2) the indexes of adjacent segments which feed into each segment, and (3) the fraction of the total flow which passes through each segment. The structure of the program allows one to alter a liquid heated garment by changing a few data statements. Hence, a situation such as loss of flow to a particular segment owing to obstruction of the tubing can be simulated with ease.

To illustrate the use of a liquid heated suit, calculations were performed for a diver wearing a 0.3 in. thick foamed neoprene dry suit over a liquid heated garment having a thickness of 1/8 inch. In some cases the breathing gas passes through a regenerative heat exchanger which produces a 25°C difference between the temperature of the supply tank and the temperature at the mouth. Humidification of the inspired gas also occurs in the heat exchanger. When the regenerator is not in the circuit, the temperature and dew point of the inspired gas are set equal to the water temperature. Temperatures and heat transfer rates were computed for a 50 minute exposure to 0°C water at a pressure of 10 ata. Computed results are summarized in Table 1.

Table 1. Summary of Results for Simulated Dives at 10 Ata.

Dives	Init. Cond.	406	403	353	396	394	401
Q_{MET}	--	6.00	6.00	6.00	1.82	1.82	1.82
W_{LIQ}	--	0.076	0.379	0.076	0.076	0.227	0.379
T_{INL}	--	44.44	44.44	44.44	44.44	44.44	44.44
ΔT_{REG}	--	0	0	25	25	25	25
T_{RE}	37.01	37.10	37.72	37.04	36.31	36.52	36.70
T_{BR}	37.00	36.39	37.24	37.14	36.17	36.44	36.67
T_{CH-S}	34.92	32.25	35.90	31.53	31.64	34.06	35.32
T_{H-S}	35.02	35.74	38.01	36.39	35.61	36.81	37.72
T_{CA-S}	34.01	24.09	29.34	24.54	23.02	25.55	27.91
Q_{RES}	--	3.84	2.68	0.83	0.79	0.57	0.33
Q_{ENV}	--	7.64	9.29	7.60	7.46	8.41	9.10
Q_{SHIV}	--	3.44	-0.38	-0.07	4.20	2.54	1.04
Q_{LHG}	--	1.51	5.90	1.50	1.56	4.30	6.21

1. All Q's are measured in Kcal/min.
2. All T's are measured in °C.
3. W is measured in kgm/min.
4. Notation -
   - Q_{MET} = metabolic rate not including shivering
   - W_{LIQ} = flow rate through liquid heated garment
   - T_{INL} = inlet temperature to liquid heated garment
   - ΔT_{REG} = temperature rise across regenerative heat exchanger
   - T_{RE} = rectal temperature
   - T_{BR} = temperature of brain
   - T_{CH-S} = temperature of chest skin
   - T_{H-S} = temperature of head skin
   - T_{CA-S} = temperature of calf skin
   - Q_{RES} = rate of heat loss through the respiratory tract
   - Q_{ENV} = rate of heat loss by convection
   - Q_{SHIV} = rate of heat generation due to shivering (negative values denote sweat production, without evaporation)
   - Q_{LHG} = rate of heat input by liquid heated garment

The results illustrate several points. One is that this environment presents a severe thermal stress for the diver. The rate of heat transfer to the environment depends on the rate at which warm water circulates through the liquid heated garment, but it is never less than 7 Kcal/min for the cases that were studied. Therefore, auxiliary heating is required, except when the diver is working vigorously (metabolic rate = 6 Kcal/min). Even for the working diver, heat loss through the respiratory system must be limited if he is to maintain his thermal balance.

Calculations performed assuming that the breathing circuit contains a regenerative heat exchanger show that this is a very valuable device. It is capable of reducing the rate of heat loss through the respiratory system for the working diver from 2.6 Kcal/min to 0.8 Kcal/min. This is especially valuable because heat lost through the respiratory tract comes from deeper tissues which are well perfused with blood. Furthermore, the device is passive and requires no heat input, although it might be worthwhile to add heat if the device were to be used at greater depths.

When heat loss through the respiratory system is not restricted, it appears that a considerable difference may develop between rectal and brain temperatures. Indeed, the rectal temperature may increase slightly owing to heating by the venous return from working leg muscles while the temperature of the brain decreases owing to loss of heat through the respiratory tract. Experimental confirmation of this possibility would lend credence to the model.

Finally, it should be noted that some form of thermal control will be required for the liquid heated garment. One can vary either the flow rate or temperature of the incoming

hot water, but only variation of flow rate was investigated in this series of calculations. The inlet temperature was held constant at 44.4°C.

When the diver works at a metabolic rate of 6 Kcal/min, he will be comfortable with very little auxiliary heating. Indeed, a water flow rate of 0.076 Kgm/min produces some sweating, which is undesirable. However, the resting diver will cool rapidly under the same conditions. When the metabolic rate is reduced to 1.82 Kcal/min, central temperatures begin to decline; and after 10 minutes, the diver is shivering. Fifty minutes of exposure results in a 0.83°C fall in brain temperature, which causes severe shivering. If the flow rate of hot water is increased to 0.38 Kgm/min, 6.21 Kcal/min are released by the liquid heated garment. This is 4.7 Kcal/min greater than the rate of heat transfer at the lower flow rate. However, the rate of heat loss to the environment also increases from 7.5 to 9.1 Kcal/min. Hence, the thermal load on the diver is reduced by approximately 3 Kcal/min. His central temperature still falls and he still shivers, but not as severely as at the low flow rate.

One could conceivably develop a passive suit that provides adequate thermal protection for the resting diver, but such a suit would cause the working diver to sweat. Vasomotor control alone does not allow man to adapt to a wide range of conditions, and other mechanisms must be invoked. For the diver this could be control of the flow rate to the liquid heated garment.

CONCLUSIONS

Even though there are gaps in our knowledge of the human thermal system, it is possible to construct a mathematical model which describes many important features of thermal response in man. By combining a reasonable physiological model with conventional engineering analysis, it appears that one can simulate the thermal behavior of divers under a variety of conditions. The next step is to use the model under carefully controlled conditions so that shortcomings can be corrected and confidence developed in the model. If this can be accomplished, our ability to design improved diving systems should be greatly enhanced.

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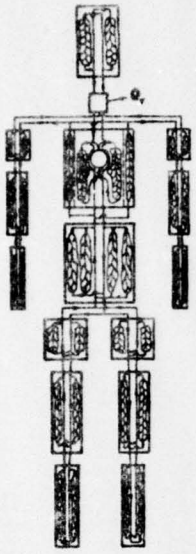


Fig. 1. Schematic diagram showing the geometric arrangement of major elements and the circulatory system.

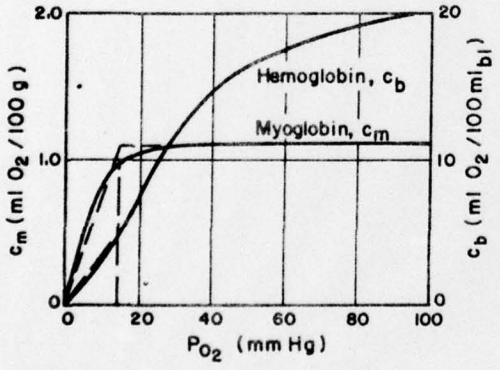


Fig. 2. Dissociation curves for myoglobin and hemoglobin.

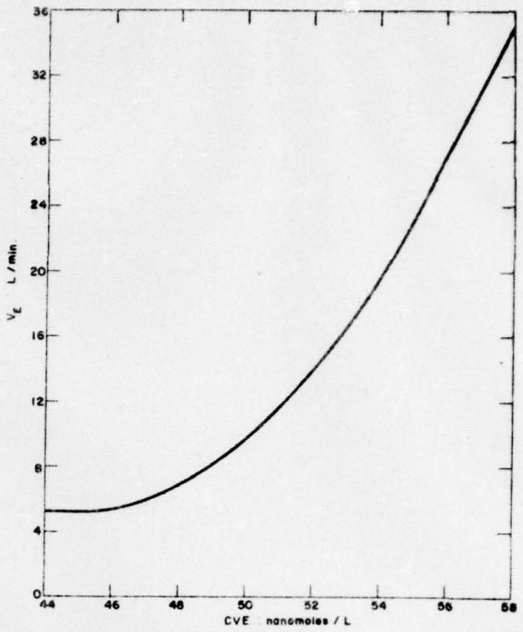
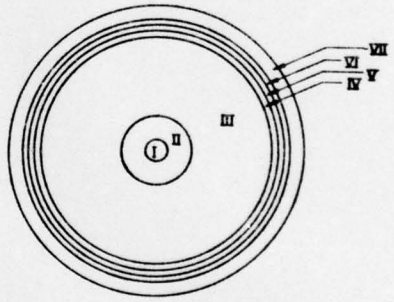


Fig. 3. Relationship between the ventilation rate and the integrated signal from chemoreceptors.



I . LARGE ARTERIES AND VEINS
II . TISSUE , SMALLER ARTERIES AND VEINS
III . MUSCLE
IV . SUBCUTANEOUS FAT AND SKIN
V . LIQUID HEATED GARMENT
VI . GAS SPACE
VII . INSULATING OUTER GARMENT

Fig. 4. Typical arrangement of radial regions in a major element such as the forearm.

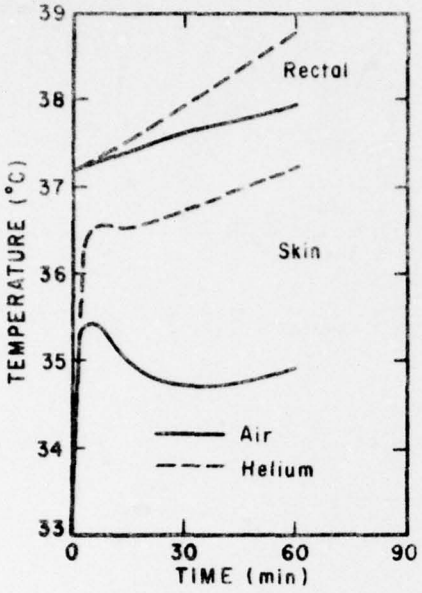


Fig. 5. Computed rectal and mean skin temperatures during exposure to 1 ata air and 7 ata helium at 41°C.

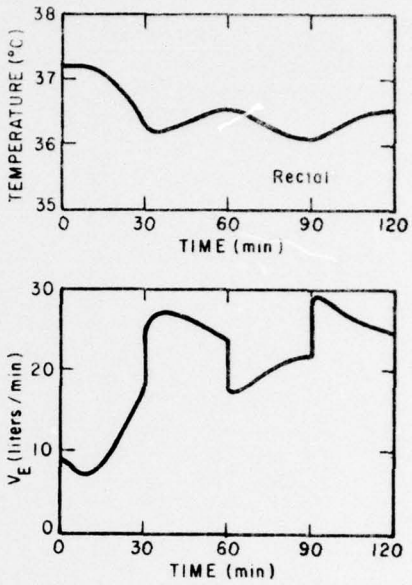


Fig. 7. Computed rectal temperature and minute volume during a shallow dive in water at 4°C with two 30 minute exercise periods.

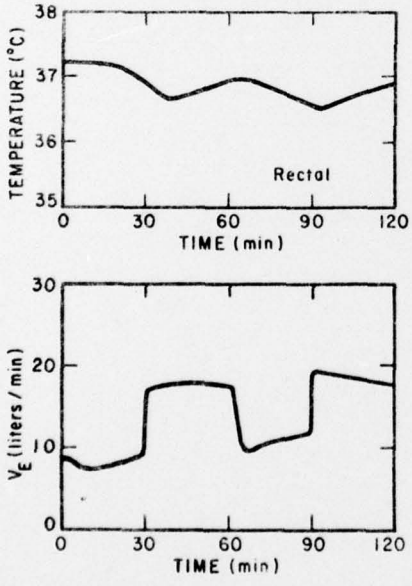


Fig. 6. Computed rectal temperature and minute volume during a shallow dive in water at 16°C with two 30 minute exercise periods.

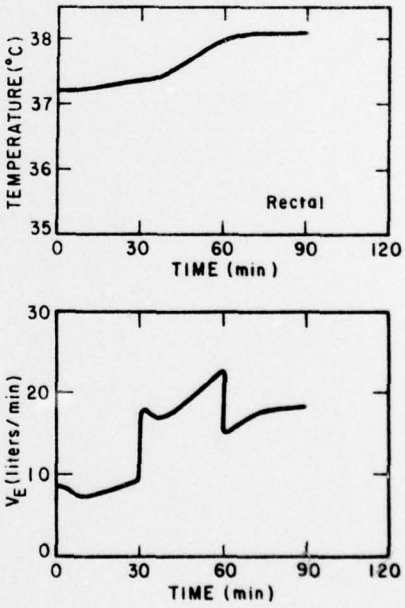


Fig. 8. Computed rectal temperature and minute volume during a shallow dive in water at 27°C with one 30 minute exercise period.

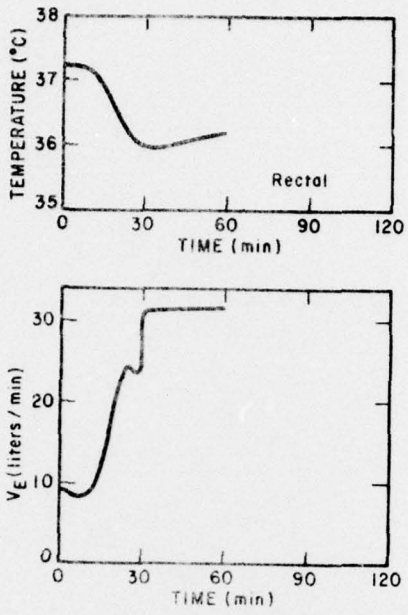


Fig. 9. Computed rectal temperature and minute volume during a dive to 30 meters in 16°C water with one 30 minute exercise period.

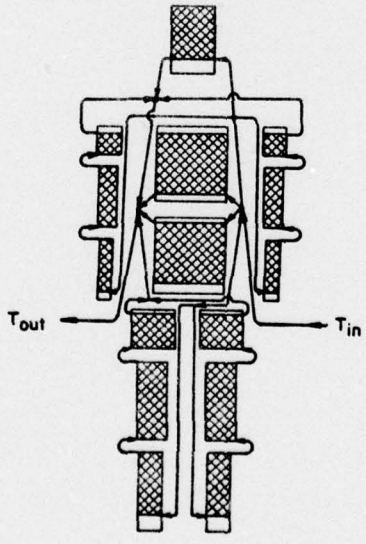


Fig. 10. Schematic representation of a typical liquid heated garment.

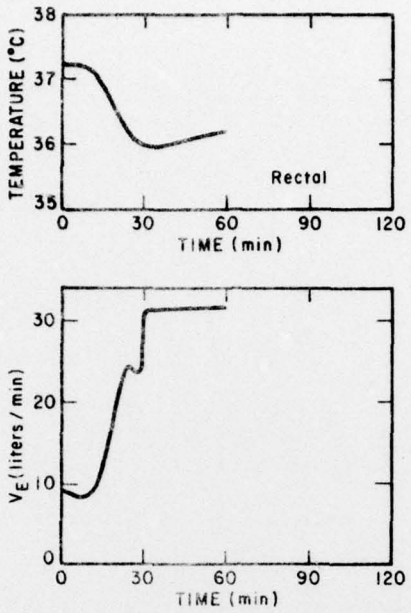


Fig. 9. Computed rectal temperature and minute volume during a dive to 30 meters in 16°C water with one 30 minute exercise period.

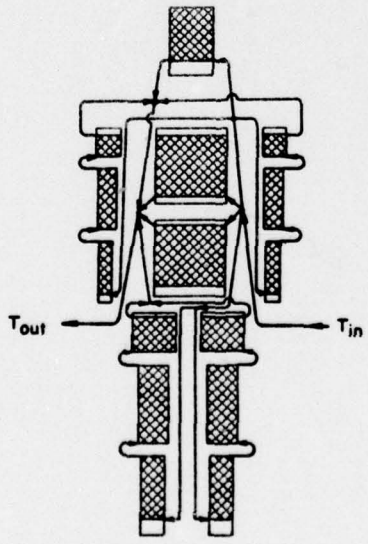


Fig. 10. Schematic representation of a typical liquid heated garment.